Fuer: WST Von: HLA Datum: 17.JUN.94
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File-Nr.	Name	Zeitraum:	Bemerkungen:
File 155:	MEDLINE(R)	1966-1994/Aug W1	
File 5:	BIOSIS PREVIEWS(R)	1969-1994/Jun ISS 21	
File 73:	EMBASE	1974-1994/ISS 24	

Suchtabelle (Suche in allen Files gleichzeitig):

S1	611	NICOTINE AND BODY (W) WEIGHT
S2	196	S1 AND PY>1989
S3	110	RD S2 (unique items)
S4	143	S1 AND PY>1990
S5	89	RD S4 (unique items)

AUSDRUCK

S/AU,TI,SO,AB/1 (Item 1 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

[Prevalence of hypertension in childhood. A study of 21 family physician practices]
Prevalenz der Hypertonie im Kindesalter. Eine Untersuchung in 21 hausaerztlichen Praxen.
Klimm HD; Reuter-Kuhn I
Fortschr Med feb 20 1994, 112 (5) p49-52,
BACKGROUND INFORMATION: Very few studies have been published on the incidence of hypertension in childhood and adolescence, in particular in the doctor's office. METHOD: In view of this, the Lehrauftrag Allgemeinmedizin at the University of Heidelberg carried out a study in the Northern part of the German State of Baden Wurttemberg involving 21 general practices and 710 children aged between 1 and 18 years. In addition to a questionnaire covering personal, social and family data, the systolic and diastolic blood pressures were determined by automatic oscillometric measurement, and also using a mercury sphygmomanometer. RESULTS: Hypertension was found in 2.3% of the children examined. At the time of diagnosis, half of all the children were suffering from some acute disease; however, more than 10% were discovered "by accident". Interesting relationships were found between hypertension, body weight, nicotine and alcohol abuse, sporting activities and family data, which, however, owing to the small number of subjects, were not amenable to more detailed analysis.

S/AU,TI,SO,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Chronic nicotine intake decelerates aging of nigrostriatal dopaminergic neurons.
Prasad C; Ikegami H; Shimizu I; Onaivi ES
Life Sci 1994, 54 (16) pl169-84,
We have evaluated the effect of chronic nicotine intake on the age-associated loss of nigrostriatal dopaminergic neurons. The striatal density of dopamine (D1 and D2) receptors and DA uptake sites decreased with age. Concomitant with these changes was a pronounced loss of many

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behavioral functions associated, at least in part, with dopaminergic neurotransmission. Chronic oral intake of nicotine resulted in partial restoration of the loss of receptors as well as behavioral performances. The results suggest that low doses of nicotine could have beneficial effects during aging.

5/AU,TI,SO,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Effect of nicotine on sympathetic nervous system activity of mice subjected to immobilization stress.
Yoshida T; Sakane N; Umekawa T; Kondo M
Physiol Behav Jan 1994, 55 (1) p53-7,
We tested the hypothesis that nicotine possesses an antistress action by measuring the turnover of norepinephrine (NE), a reliable indicator of sympathetic nervous system activity, in the interscapular brown adipose tissue (IBAT) and heart of mice treated with nicotine injections, immobilization stress, or nicotine + immobilization stress, vs. untreated controls. Nicotine enhanced the sympathetic activity in the IBAT and heart, and induced a loss of body weight. Immobilization-related stress accelerated sympathetic activity in the IBAT and heart more strongly than did nicotine loading alone. Pretreatment with nicotine suppressed the sympathetic activity induced by immobilization stress to the same extent as that observed after administering nicotine alone. Thus, these results appeared to support our hypothesis.

5/AU,TI,SO,AB/4 (Item 4 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Transdermal nicotine patches with low-intensity support to aid smoking cessation in outpatients in a general hospital. A placebo-controlled trial.
Foulds J; Stapleton J; Hayward M; Russell MA; Feyerabend C; Fleming T; Costello J
Arch Fam Med Apr 1993, 2 (4) p417-23,
OBJECTIVE: To assess whether transdermal nicotine patches combined with low-intensity support can help outpatients in a general hospital stop smoking. DESIGN: Randomized, double-blind, placebo-controlled trial with 12 weeks of follow-up. SETTING: Department of Thoracic Medicine in an inner-city public general hospital, London, England. SUBJECTS: Two hundred forty-eight outpatients in a general hospital, who smoked at least 10 cigarettes per day (the majority were being treated for smoking-related diseases), referred by clinicians at the hospital. INTERVENTION: Brief advice to stop smoking and daily application of transdermal nicotine patches (delivering 15 mg over 16 hours) or placebo, with follow-up appointments at 1, 3, 6, and 12 weeks, with a doubling of the dosage for continuing smokers at week 1. MAIN OUTCOME MEASURE: Sustained abstinence from tobacco from week 3 to week 12 validated with measurement of expired-air carbon monoxide concentration at weeks 3, 6, and 12. RESULTS: Twenty-nine (23.4%) of 124 subjects assigned to the nicotine group were validated as having abstained from smoking at both weeks 3 and 6, compared with 16 (12.9%) of 124 subjects receiving placebo (P = .008). At week 12, 22 (17.7%) of the subjects in the nicotine group were validated as having abstained at all three points as were 15 (12.1%) of the subjects in the placebo group (P = .058). CONCLUSION: Transdermal nicotine patches combined with low intensity support are effective in helping outpatients in a general hospital stop smoking but do not prevent relapse after 6 weeks.

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5/AU,TI,SO,AB/5 (Item 5 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Two studies of the clinical effectiveness of the nicotine patch with different counseling treatments.

Fiore MC; Kenford SL; Jorenby DE; Wetter DW; Smith SS; Baker TB

Chest Feb 1994, 105 (2) p524-33,

OBJECTIVE: To assess the effectiveness of transdermal nicotine therapy for smoking cessation and suppression of withdrawal severity in conjunction with two different adjuvant counseling treatments. DESIGN: Two independent randomized placebo-controlled double-blind trials. SETTING: Smoking cessation clinic. SUBJECTS: Eighty-eight (study 1) and 112 (study 2) adult volunteers motivated to quit smoking. INTERVENTIONS: Eight weeks of 22-mg transdermal nicotine therapy with group counseling (study 1); 4 weeks of 22 mg followed by 2 weeks of 11-mg transdermal nicotine therapy with brief individual counseling (study 2). MAIN OUTCOME MEASURES: Modified point prevalence (7 consecutive days of nonsmoking) at the end of patch treatment and 6 months after treatment initiation was assessed by self-report and biochemically confirmed; survival analyses were also conducted for both studies to compare treatment efficacy. Also, we examined the impact of the nicotine patch on specific withdrawal symptoms (anger, anxiety, awakening, difficulty concentrating, depression, hunger, impatience, and craving). RESULTS: Transdermal nicotine treatment produced higher cessation rates at the end of treatment than did placebo with both adjuvant counseling interventions: 59 percent vs 40 percent ($p < 0.05$ in study 1) and 37 percent vs 20 percent ($p < 0.05$ in study 2), respectively. Smoking cessation efficacy was maintained 6 months after initiation of treatment: 34 percent vs 21 percent ($p = 0.08$ in study 1) and 18 percent vs 7 percent ($p = 0.05$ in study 2). Survival analyses also revealed significant group differences in efficacy in both studies. Nicotine patches also suppressed a variety of withdrawal symptoms, including craving in the first weeks after patients quit smoking. CONCLUSION: Transdermal nicotine effectively augments smoking cessation rates with two different types of counseling treatment. Overall, the nicotine patch approximately doubles the sustained rate of smoking cessation. Additionally, the nicotine patch provides relief from some tobacco withdrawal symptoms.

5/AU,TI,SO,AB/6 (Item 6 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Effects of snuff on regional blood flow to the cheek and tongue of anesthetized dogs.

Huckabee KD; Barnes RT; Williams AG Jr; Fan WL; Downey HF

Oral Surg Oral Med Oral Pathol Dec 1993, 76 (6) p729-35,

Regional blood flow to the cheek and tongue of anesthetized dogs was measured before and after application of six different doses of snuff (3.12 to 100 mg/kg body weight) to the right cheek. Dose-independent vasodilation was observed at the site of application, whereas dose-dependent vasoconstriction was observed contralaterally. Aortic, central venous, and ventricular pressures were significantly increased ($p < 0.05$) at the higher doses. Plasma nicotine concentrations increased stepwise with dose.

5/AU,TI,SO,AB/7 (Item 7 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Expression of prostaglandin E2 receptor in hamster buccal pouch: effect of benzo (a) pyrene and nicotine.

Wu-Wang CY; Wang SL; Brigham M; Correa A; Slomiany A; Slomiany BL

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Source: https://www.industrydocuments.ucsf.edu/docs/xndl0000

Prostaglandins Leukot Essent Fatty Acids Oct 1993, 49 (4) p821-6,

Prostaglandin E2 (PGE2) plays an important role in the maintenance of oral mucosal integrity. In this study, we characterized PGE2 receptor binding in the buccal mucosa of Syrian hamster and assessed the effect of nicotine (NC) and benzo (a) pyrene (BP), the two major ingredients in cigarette smoke, on this receptor. Adult male animals were treated for 4 weeks by apical swabbing of the buccal pouch with corn oil (control, C), 1 mM NC, BP, or NC + BP in corn oil, twice a day, 5 days a week. The results obtained with the untreated group revealed the presence of a specific PGE2 receptor consisting of two binding sites (high affinity with Kd = 1.52 nM and Bmax = 37 fmol/mg protein and low affinity with Kd = 813 nM and Bmax = 1.29 pmol/mg protein). The treatment with NC, BP, and NC + BP caused a significant decrease in PGE2 receptor binding (specific binding: 10.20 +/- 0.42, 6.84 +/- 1.32**, 6.58 +/- 0.67** and 5.88 +/- 1.03** fmol/mg protein; C, NC, BP, and NC+BP, respectively; Mean +/- SD, n = 5, **p < 0.01). The data suggest that decreased receptor binding for PGE2 in the buccal mucosa may be the cause for the adverse effect of cigarette smoke on the health of oral mucosa.

5/AU,TI,SO,AB/8 (Item 8 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Effects of nicotine cycling on weight loss and regain in male rats.
Winders SE; Wilkins DR 2d; Rushing PA; Dean JE
Pharmacol Biochem Behav Sep 1993, 46 (1) p209-13,

Over successive periods of weight loss and regain caused by deprivation and refeeding, weight loss becomes slower during deprivation and weight is regained quicker during refeeding. One period of nicotine administration and termination results in changes in intake and weight gain comparable to that caused by one period of food deprivation and refeeding. However, no study has examined the effect of multiple periods of nicotine administration and cessation on weight loss and regain. The present study examined the effect of repeated cycles of nicotine administration and cessation on growth rate. Thirty two male Sprague-Dawley rats underwent three nicotine administration cycles. Cycles consisted of 2 weeks of nicotine or saline followed by 2 weeks of no drug. Nicotine administration decreased growth and food consumption, and cessation resulted in a resumption of normal growth and intake. However, changes in food consumption and body weight were similar across cycles. Thus, although nicotine administration and cessation produced reliable changes in food consumption and body weight similar to those caused by diet cycling, there were no comparable cumulative effects of nicotine cycling on growth rate.

5/AU,TI,SO,AB/9 (Item 9 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Effectiveness of a 16-hour transdermal nicotine patch in a medical practice setting, without intensive group counseling.
Sachs DP; Sawe U; Leischow SJ
Arch Intern Med Aug 23 1993, 153 (16) p1881-90,

BACKGROUND: To determine the effectiveness of a 16-hour transdermal nicotine patch in assisting smokers to stop smoking, when used in a primary medical practice model. METHODS: A single-site, randomized, double-blind, out-patient, parallel-group, placebo-controlled trial consisting of 220 regular, otherwise healthy cigarette smokers. Patients participated in a 12-week patch treatment phase plus a 6-week tapering phase. A standard medical office model of physician intervention, such as could easily be employed by any primary care physician, without need for any special

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psychological services, training, or skills, was the behavioral intervention. RESULTS: Sustained abstinence, determined at each visit by absolutely no cigarette use, carbon monoxide level of 9 ppm or less, and serum cotinine level of 15 ng/mL or less (after week 18), was significantly greater for those patients receiving the active nicotine patch than for those receiving the placebo patch: the percent of patients not smoking at 6, 12, 18, 26, and 52 weeks was 61% vs 35%, 45% vs 26%, 41% vs 16%, 34% vs 12%, and 25% vs 9%, respectively ($P < .001$). This 16-hour nicotine patch produced no systemic side effects and minimal skin irritation. CONCLUSIONS: Nicotine replacement therapy via a 16-hour transdermal nicotine patch provided safe and effective treatment for tobacco-dependent patients. One-year sustained nonsmoking rates were nearly three times higher in the active than in the placebo condition, when the patch was used in an easily applicable standard medical practice setting, without the need for psychological interventions. This outcome was as good as or better than results achieved by nicotine patches using behavior modification or group counseling.

5/AU,TI,SO,AB/10 (Item 10 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Weight gain following smoking cessation.
Perkins KA
J Consult Clin Psychol Oct 1993, 61 (5) p768-77,

Smoking generally suppresses body weight below "normal," and smoking cessation allows weight to return to normal. This weight gain following cessation appears to be due to a transient increase in eating coupled with the removal of acute metabolic effects of each cigarette, with no change in physical activity. Nevertheless, tobacco smoke (and specifically nicotine) does not appear to be simply either an anorectic or a thermogenic agent. Although there may be no easy explanation for the effects of smoking on energy balance, the most parsimonious explanation may be that smoking lowers body weight "set point" and cessation raises set point. The transient changes in eating are therefore secondary to the changes in body weight set point. This notion is supported by animal research with nicotine as well as with other drugs, and it is also supported less directly by the pattern of changes observed with changes in smoking status among humans. A set-point explanation for weight gain after smoking cessation may also help explain the lack of success of interventions designed to prevent this weight gain.

5/AU,TI,SO,AB/11 (Item 11 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Increased aortic endothelial death and enhanced transendothelial macromolecular transport in streptozotocin-diabetic rats.
Lin SJ; Hong CY; Chang MS; Chiang BN; Chien S
Diabetologia Oct 1993, 36 (10) p926-30,

Hypertension, cigarette smoking and diabetes mellitus are well-known risk factors for atherosclerosis and coronary heart disease. Repeated endothelial cell injury and increased lipid entry have been suggested as initiating events in atherogenesis. Our previous studies have demonstrated that the frequency of endothelial cell death and associated endothelial permeability were significantly increased in the aorta of spontaneously hypertensive rats and chronic oral nicotine-treated rats. In the present investigation, we examined the hypothesis that diabetes also increases the frequency of arterial endothelial cell death and hence transendothelial macromolecular transport, which may have some implications in increasing

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lipid entry and thus accelerating atherogenesis. Diabetes was induced in 15 male Sprague Dawley rats by intraperitoneal injection of 60 mg streptozotocin per kg body weight. The duration of diabetes was 6 weeks. A group of 15 age matched rats, injected only with the buffer and maintained over the same time period, served as the controls. In en face preparations of the thoracic aorta, IgG-containing dead endothelial cells were identified by an indirect immunoperoxidase method, and endothelial leakage to Evans blue-albumin complexes was quantified by fluorescence microscopy. Diabetic rats, compared to control rats, had significantly higher values for the frequency of endothelial cell death (0.77 +/- 0.10% vs 0.38 +/- 0.04%; p < 0.005 by two tailed, unpaired Student's t-test) and the number density of Evans blue-albumin leaky foci (4.33 +/- 0.48/mm2 vs 2.99 +/- 0.38/mm2; p < 0.05 by two tailed, unpaired t-test) in the aorta.(ABSTRACT TRUNCATED AT 250 WORDS)

5/AU,TI,SO,AB/12 (Item 12 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

[Tobacco and weight]
Tabac et poids.
Louis-Sylvestre J
Rev Prat May 15 1993, 43 (10) p1235-7,

The weight increase which immediately follows the cessation of smoking is often dissuasive. All things being equal otherwise, the weight and adiposity of smokers are lower than those of non-smokers. The calorie intake of smokers is equal to that of non-smokers, but the nicotine absorbed with each cigarette smoked temporarily increases the basal metabolism and in particular the energy expended in all physical activities. The lack of compensatory increase of food intake in response to the increase of energy expenditure explains why smokers are underweight. Food consumption might be reduced by an increase of circulating fatty acids and by a nicotine induced inhibition of insulin secretion. The weight increase observed after tobacco withdrawal is caused by the normalization of expenditures and by release of the brake on food consumption. Weight increase is usually important in heavy smokers, women and subjects with low physical activity. Fifty per cent of smokers who have given up smoking put on less than 3 kg. The effort required to mitigate this disadvantage is modest and it can be delayed and separated from the acute weaning period with a medicinal treatment.

5/AU,TI,SO,AB/13 (Item 13 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Prenatal nicotine exposure and cognitive performance in rats.
Levin ED; Briggs SJ; Christopher NC; Rose JE
Neurotoxicol Teratol Jul Aug 1993, 15 (4) p251-60,

In humans and animal models there is evidence that prenatal nicotine exposure causes lasting deficits in cognitive performance. The current study examined the cognitive effects of prenatal exposure of rats to 2 mg/kg/day of nicotine. This dose did not cause significant deficits in maternal weight gain, offspring litter size, or pup weight. The control offspring showed the normal ontogeny of spontaneous alternation from near chance (50%) performance to 80%-85% alternation. In contrast, the nicotine exposed rats had the opposite progression with abnormally high alternation on days 22-39 and abnormally low alternation on days 35-52. Acquisition of choice accuracy performance on the radial-arm maze (RAM) was not altered in a major way by nicotine exposure. Minor nicotine-induced changes in choice accuracy were seen during the initial trials of

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acquisition. The nicotine exposed female offspring had a significantly longer response duration. Prenatal nicotine exposure did significantly alter the effects of subsequent drug challenges on choice accuracy performance. The nicotine exposed male offspring were significantly more responsive to the amnestic effects of the nicotinic antagonist mecamylamine. In a subsequent challenge, the effects of the beta-adrenergic antagonist propranolol were examined. A significant dose-related impairment in choice accuracy was seen in the control rats. In contrast, the nicotine-exposed rats did not show any significant response to propranolol. This decreased responsiveness to adrenergic challenge parallels the reduction in adrenergic response to nicotine challenge we previously found in littermates to the rats of the current study. Prenatal nicotine exposure causes subtle alterations in cognitive performance that can be magnified by challenges of nicotinic and adrenergic systems.

5/AU,TI,SO,AB/14 (Item 14 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Nicotine pharmacokinetics of Nicoderm (nicotine transdermal system) in women and obese men compared with normal-sized men.

Prather RD; Tu TG; Rolf CN; Gorsline J

J Clin Pharmacol Jul 1993, 33 (7) p644-9,

This study examined the effect of gender and body weight on the pharmacokinetic properties of the Nicotine Transdermal System (NTS) (Nicoderm). This NTS was applied for 24 hours to 13 normal-sized men, 13 women, and 13 obese men, all of whom were smokers who had abstained from cigarettes for the previous 24 hours. Pharmacokinetic parameters were determined during a single application of the system. The mean nicotine maximal plasma concentration (Cmax) and area under the curve (AUC) values for women did not differ significantly from those for normal-sized men. Nicotine Cmax and AUC values, however, were significantly lower in obese compared with normal-sized men; nicotine AUC was strongly correlated to body weight and body mass index. Mean apparent nicotine elimination rate constant values were not significantly different between normal-sized and obese men, but the apparent elimination rate constant value was significantly higher in women. The possible clinical significance of the differences in nicotine AUC values with body weight is discussed.

5/AU,TI,SO,AB/15 (Item 15 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

The influence of maternal nicotine exposure on neonatal lung metabolism. Protective effect of ascorbic acid.

Maritz GS

Cell Biol Int Jun 1993, 17 (6) p579-85,

The aim of the present study was to establish whether ascorbic acid supplementation (1 mg/kg/body mass/day) during pregnancy and lactation will prevent the effect of maternal nicotine exposure (1 mg/kg/body weight/day) on neonatal lung carbohydrate, DNA and protein metabolism. The data show that the adult lung ascorbic acid content was reduced by 76% after exposure to nicotine. In contrast, maternal nicotine exposure during pregnancy and lactation has no effect on neonatal lung ascorbic acid content. However, ascorbic acid supplementation during pregnancy and lactation prevented the adverse effects of maternal nicotine exposure on neonatal lung carbohydrate, DNA and protein metabolism.

5/AU,TI,SO,AB/16 (Item 16 from file: 155)

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DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Behavioral effects of prenatally administered smokeless tobacco on rat offspring.

Paulson RB; Shanfeld J; Vorhees CV; Sweazy A; Gagni S; Smith AR; Paulson JO

Neurotoxicol Teratol May-Jun 1993, 15 (3) p183-92,

Two dosages of Smokeless Tobacco (ST) extract were given to gravid Sprague-Dawley rats by oral gavage on gestational days (GD) 6-20. The low dosage contained ST extract equivalent to 1.33 mg/kg nicotine (STD-1), and the high dosage contained ST extract equivalent to 4.0 mg/kg nicotine (STD-2). Dams were dosed three times daily at 8 a.m., 11 a.m., and 2 p.m., thus providing total daily nicotine equivalent dosages of 4 mg/kg/day and 12 mg/Kg/day. Controls received equivalent amounts of distilled water by gavage. Dams were allowed to deliver and all experimental pups were fostered to control mothers. On postnatal day 1 (PND 1) litters were culled to 4 +/- 1 females and 4 +/- 1 males. Weights, physical landmark development, and behavioral performance of pups were monitored during pre- and post-weaning periods. Behavioral tests included: surface righting, negative geotaxis, swimming development, open-field activity, active avoidance in shuttle box, and Cincinnati swimming maze. Our results show that the STD-2 dose resulted in reduced maternal weight gain. Offspring weights were reduced in a dose-related manner, with the most consistent weight deficits seen in the STD-2 group until PND29. Consistent STD-1 weight deficits were seen up to PND 8. The incidence of deaths was increased in the STD-2 dosage group. No significant treatment-related differences were observed in development of physical landmarks. Male STD-2 pups righted faster than controls, and significant differences were noted in swimming development with the STD-1 group of pups performing less effectively than controls. Activity levels, assessed during both pre- and post-weaning periods were not affected. No treatment-related differences were seen in the active avoidance shuttle box or Cincinnati swimming maze tests, which assessed learning. Female brain weights were reduced in the STD-1 treatment group.

5/AU,TI,SO,AB/17 (Item 17 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Weight change after smoking cessation in general practice.

Richmond RL; Kehoe L; Webster IW

Med J Aust Jun 21 1993, 158 (12) p821-2,

OBJECTIVE: To examine weight change among patients attending a smoking cessation program administered by general practitioners. DESIGN: Body weight and smoking level were measured at the initial assessment and at a six-month follow up visit. Pretreatment measures of demographic, attitudinal and smoking-related variables were examined for relationship to weight gain. PARTICIPANTS: A total of 235 patients who were part of a smoking cessation study in Sydney. MAIN OUTCOME MEASURE: Change in weight over six months. RESULTS: Abstainers (n = 52) gained an average of 4.0 kg, significantly more than continuing smokers who gained an average of 1.2 kg. Among abstainers, only self-ratings of good health predicted weight gain. CONCLUSIONS: Results indicate that weight gain among general practice patients following smoking cessation is similar to that in other research settings.

5/AU,TI,SO,AB/18 (Item 18 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

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The effect of chronic nicotine administration on bone mineral content in mice.

Broulik PD; Jarab J

Horm Metab Res Apr 1993, 25 (4) p219 21,

Tobacco use has been identified as being a risk factor for the development of osteoporosis. To elucidate the effect of nicotine on bone metabolism we examined 8 intact and 8 castrated mice treated for 56 days with nicotine in drinking water and compared with the same number of mice acting as controls. The mineral bone mass in the femora of the animals was measured quantitatively. A significant reduction of bone density and bone mineral content was found in the nicotine treated animals compared to animals without nicotine. Nicotine itself does not exert any antiandrogenic effect, and it does not produce changes in the weight of seminal vesicles.

5/AU,TI,SO,AB/19 (Item 19 from file: 155)

DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Factors influencing vaginal cytology, pH and bacterial flora in elderly women.

Milsom I; Arvidsson L; Ekelund P; Molander U; Eriksson O

Acta Obstet Gynecol Scand May 1993, 72 (4) p286-91,

OBJECTIVE. To evaluate factors influencing the vaginal micro-environment in elderly women. STUDY DESIGN. Vaginal pH, cytology (Karyopyknotic index, KPI) and bacterial cultures were assessed in 350 women (age 72.2 +/- 1.4 years, range 65-84 years) who were not taking estrogens. RESULTS. Vaginal pH was 6.5 +/- 0.1, KPI was 3.0 +/- 0.9% and bacteria of fecal type dominated. However, in some women the vaginal micro-environment resembled that of fertile women, with a predominance of lactobacilli (n = 68), low pH (n = 41) and an increased maturation index (n = 57). KPI was higher ($p < 0.001$) in women weighing > or = 85 kg, or with a body mass > or = 9th decentile or with a diastolic blood pressure > or = 100 mmHg and in women ($p < 0.05$) with a high menopausal age (> or = 53 years). Vaginal pH was lower ($p < 0.001$), KPI was higher ($p < 0.01$) and there was an increased ($p < 0.05$) presence of lactobacilli in smokers compared to non-smokers. CONCLUSIONS. The latter finding is somewhat paradoxical as cigarette smoking has previously been shown to negatively influence estrogen metabolism. The influence of cigarette smoking on the vaginal micro-environment may be a direct local effect of nicotine.

5/AU,TI,SO,AB/20 (Item 20 from file: 155)

DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Body fatness and smoking history predict the thermic effect of smoking in fasted men.

Walker JF; Collins LC; Vogel RL; Stamford BA

Int J Obes Relat Metab Disord Apr 1993, 17 (4) p205-8,

Epidemiological studies suggest that body composition and smoking history are related to degree of weight gain following smoking cessation. We hypothesized that body composition and smoking history affect the thermic effect of smoking (TES), which in turn would influence weight gain. Forty males ranging in age from 20 to 70 years smoked two cigarettes (0.8 mg nicotine yield) in 20 minutes, after which resting energy expenditure (REE) was measured during the next hour. The average change in REE (delta REE) was 4.97% ($P < 0.0001$), with a range of -2% to +14%. delta REE was greater than 1% in 35 subjects, less than 1% in three subjects, and reduced in two subjects. Body fatness was negatively correlated with delta REE ($r = -0.68$, $r2 = 0.46$). Multiple regression analysis indicated that body fatness (%) and pack-year history of smoking strongly predict delta REE ($R = 0.82$, $R2 =$

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Ø.68; delta REE = 11.ØØØ-Ø.296 x %FAT-Ø.Ø37 x pack-year). It was concluded that body fatness and smoking history substantially influence the thermic effect of smoking. In addition, this finding helps explain the wide range in TES which has been reported among subjects in many previous studies.

5/AU,TI,SO,AB/21 (Item 21 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Impact of fetal nicotine exposure on development of rat brain regions: critical sensitive periods or effects of withdrawal?
Slotkin TA; Lappi SE; Seidler FJ
Brain Res Bull 1993, 31 (3-4) p319-28,
Fetal nicotine exposure evokes alterations in central nervous system structural, neurochemical, and behavioral development. In the current study, the relative importance of critical developmental exposure periods and withdrawal were examined by infusing nicotine to pregnant rats using osmotic minipumps beginning on the fourth day of gestation. Infusions were confined to either the first 8 days (withdrawal on gestational day 13), to nearly all of gestation (withdrawal on gestational day 21), or throughout gestation and continued into the first 2 postnatal weeks. Maternal weight gain was retarded by nicotine, with a hierarchy corresponding to the duration of nicotine exposure. Similarly, fetal and neonatal body weights were unaffected in the group receiving the shortest duration of nicotine exposure, and were less affected by the intermediate infusion regimen than by the longest regimen; brain region weights were reduced significantly only with the longest regimen. Using ODC activity, a sensitive marker for altered brain cell development, we found little change in animals exposed to nicotine in early gestation and undergoing withdrawal on day 13. However, in the groups receiving nicotine through the end of gestation or through gestation and into the postnatal period, ODC activity was significantly elevated. These results indicate that withdrawal from nicotine contributes little, if any, effect either to the growth deficits or to abnormalities of brain cell development. Instead, the most important factor appears to be exposure within the developmental period corresponding to the proliferation of nicotinic receptors and the timing of receptor control of cell replication and differentiation.

5/AU,TI,SO,AB/22 (Item 22 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Nicotine and cotinine elimination pharmacokinetics in smokers and nonsmokers.
Benowitz NL; Jacob P 3d
Clin Pharmacol Ther Mar 1993, 53 (3) p316-23,
Published research suggests that smokers metabolize nicotine and its major metabolite cotinine more rapidly than nonsmokers. To evaluate this possibility, we studied the disposition pharmacokinetics of intravenous deuterium-labeled nicotine in 11 smokers (administered 2.Ø and Ø.5 micrograms/kg/min x 3Ø minutes) and in 11 nonsmokers (Ø.5 micrograms/kg/min). Smokers did not metabolize nicotine more rapidly; the clearance of nicotine normalized for body weight was significantly slower in smokers than in nonsmokers. Cotinine elimination rates were similar in the two groups. The disposition pharmacokinetics of nicotine was similar for the low and high doses in the smokers, indicating that metabolism is dose-independent. Our findings argue against "metabolic tolerance" as a mechanism for the dose escalation observed in smokers after initiation of smoking. Our findings suggest that nicotine and cotinine kinetic parameters for smokers may be extrapolated to nonsmokers for estimating exposures to

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environmental tobacco smoke in nonsmokers.

5/AU,TI,SO,AB/23 (Item 23 from file: 155)
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Sertraline attenuates hyperphagia in rats following nicotine withdrawal.
Levin ED; Briggs SJ; Christopher NC; Rose JE
Pharmacol Biochem Behav Jan 1993, 44 (1) p51-61,
Chronic nicotine administration can decrease food consumption and body weight. Abrupt withdrawal from nicotine can cause the reverse effect, hyperphagia and rapid weight gain. In the current study, the efficacy of sertraline, a serotonin reuptake inhibitor, on nicotine withdrawal-induced hyperphagia and rapid weight gain was assessed in rats. Sertraline was found to be effective in reversing the increase in feeding that occurred after withdrawal from chronic nicotine administration. Sertraline caused a dose-related decrease in food consumption in control rats not given nicotine. Doses of 5 and 10 mg/kg/day caused significant decreases while 2.5 mg/kg/day caused a slight though nonsignificant decrease in food consumption. Rats in which nicotine was abruptly withdrawn after 3 weeks of administration showed a significant increase in food consumption relative to controls. This increase was eliminated by the high dose of sertraline (10 mg/kg/day), but not by the lower two doses (2.5 and 5 mg/kg/day). Water consumption was affected in a similar fashion. Body weight gain was also affected by sertraline. During the first week after nicotine withdrawal, rats rapidly gained weight, but sertraline attenuated this. The 10-mg/kg dose of sertraline significantly attenuated the nicotine withdrawal-induced weight gain. These results suggest that sertraline can counteract the hyperphagia and rapid weight gain associated with nicotine withdrawal, and might therefore be a useful adjunct to smoking cessation.

5/AU,TI,SO,AB/24 (Item 24 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Sensitivity of lateral hypothalamic neurons to nicotine: origin and possible correlation with nutritional effects of nicotine.
Himmi T; Brahiti F; Perrin J; Orsini JC
Pharmacol Biochem Behav Jan 1993, 44 (1) p217-20,
Single-unit activity was recorded extracellularly in the lateral hypothalamus of anesthetized rats. A number of neurons responded to intravenous nicotine, but most failed to respond similarly to local nicotine or systemic administration of a peripheral acting agonist. This finding suggests that these neurons respond indirectly to systemic nicotine through afferent pathways originating in central nicotinoceptive cells. The incidence of response was significantly greater in the cells sensitive to moderate changes in blood glucose. This finding suggests that the effects of peripheral nicotine on food intake and body weight are partly mediated by "glycemia-sensitive neurons" in the lateral hypothalamus.

5/AU,TI,SO,AB/25 (Item 25 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Smoking rate, carboxyhemoglobin, and body mass in the Second National Health and Nutrition Examination Survey (NHANES II).
Istvan JA; Cunningham TW
J Behav Med Dec 1992, 15 (6) p559-72,
The relationship of body mass and triceps skinfold thickness to both reported number of cigarettes smoked per day and carboxyhemoglobin levels

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was examined in healthy cigarette smokers in the NHANES II. Among both men and women, higher carboxyhemoglobin levels were related to lower body mass and thinner skinfolds, whereas higher levels of reported daily cigarette smoking were related to increased body mass and thicker skinfolds among men only. These relationships were independent of age, education, caloric intake, physical activity, and exercise. The opposite effects of number of cigarettes smoked per day and a biological index of cigarette smoke exposure on body mass suggest that increased cigarette smoking may covary with factors that would favor increased body weight among men, whereas decreases in body weight with increases in carboxyhemoglobin may reflect the effects of nicotine exposure on energy expenditure in both men and women.

5/AU,TI,SO,AB/26 (Item 26 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Fetal hemodynamic response to maternal intravenous nicotine administration.
Clark KE; Irion GL
Am J Obstet Gynecol Dec 1992, 167 (6) p1624-31,
OBJECTIVES: Our study was designed to test the hypothesis that maternally administered nicotine has significant effects on fetal hemodynamics and umbilical systolic/diastolic ratios. STUDY DESIGN: Nine pregnant ewes received maternal intravenous infusions of 10, 20, and 30 micrograms/kg/min of nicotine. Maternal and fetal blood pressure, heart rate, and uterine and umbilical blood flow were recorded. RESULTS: Maternal intravenous administration of nicotine (10, 20, and 30 micrograms/kg/min of maternal body weight) produced significant ($p < 0.05$) increases in fetal blood pressure (2%, 11%, and 25%, respectively), decreases in fetal heart rate (0%, 8%, and 12%), and decreases in umbilical blood flow (0%, 0%, and 19%). Umbilical systolic/diastolic ratios increased slightly at the 30 micrograms/kg/min dose of nicotine, but these changes did not reach significance. Maternal blood pressure increased (10%, 25%, and 53%), and uterine vascular resistance increased (5%, 64%, and 344%) significantly ($p < 0.05$); uterine blood flow increased at the 10 micrograms/kg/min dose (+5%) and decreased by 23% and 42% at the highest two doses of nicotine. CONCLUSION: Maternal nicotine administration in late-term pregnant sheep produced significant increases in fetal arterial blood pressure and umbilical vascular resistance, decreased fetal heart rate, and umbilical blood flow but did not significantly alter systolic/diastolic ratios.

5/AU,TI,SO,AB/27 (Item 27 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Rodent model of nicotine abstinence syndrome.
Malin DH; Lake JR; Newlin-Maultsby P; Roberts LK; Lanier JG; Carter VA; Cunningham JS; Wilson OB
Pharmacol Biochem Behav Nov 1992, 43 (3) p779-84,
Few animals models are currently in use for the recognized clinical problem of nicotine dependence and abstinence. This study introduces a rapid and convenient model using the rat. Sixteen male rats were rendered nicotine dependent by 7 days of continuous subcutaneous infusion of either 3 mg/kg/day (n = 8) or 9 mg/kg/day (n = 8) nicotine tartrate salt; 8 control rats were infused with saline alone. Rats were observed for 15 min before, during, and after the drug infusion period using a tally sheet modified from a standard checklist of opiate abstinence signs. There were few signs observed in any group at baseline and at the end of the infusion period. However, nicotine-infused rats showed a significant, dose-related

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increase over the control group at 16 h after the end of infusion, largely subsiding by 10 h. The most frequently observed signs during withdrawals included: teeth-chattering/chews, writhes/gasps, ptosis, tremors/shakes, and yawns. A significant drop in locomotor activity and increase in weight gain following termination of nicotine infusion provided additional evidence of an abstinence syndrome. This syndrome was alleviated by SC administration of 0.4 mg/kg nicotine tartrate.

5/AU,TI,SO,AB/28 (Item 28 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

A study of tobacco carcinogenesis XLVIII. Carcinogenicity of N'-nitrosonornicotine in mink (Mustela vison).
Koppang N; Rivenson A; Reith A; Dahle HK; Evensen O; Hoffmann D
Carcinogenesis Nov 1992, 13 (11) p1957-60,
During tobacco processing and smoking, nicotine and nornicotine give rise to N'-nitrosonornicotine (NNN), a highly abundant, strong carcinogen. NNN is known to exert carcinogenic activity in mice, rats and hamsters. Major target organs for NNN carcinogenicity in the rat are the esophagus and the nasal mucosa, and in the Syrian golden hamster trachea and nasal mucosa. In comparison with the rat, the mink (Mustela vison) has a markedly expanded nasal mucosa. Therefore, we explored in this study whether the mink could serve as a non-rodent model for nasal carcinogenesis using NNN as the carcinogen. Twenty random-bred mink, beginning at the age of 3 weeks, received twice weekly s.c. injections of NNN, a total dose of 11.9 mM per animal over a 38 week period. All of the 19 mink at risk developed malignant tumors of both the respiratory and the olfactory region of the nose within 3.5 years. In most animals the malignant tumors, primarily esthesioneuroepithelioma, invaded the brain. Remarkably, NNN induced no other tumors in the mink. None of the control animals developed nasal tumors nor tumors at other sites during the 3.5 years of the assay. The historical data from the farm did not reveal any spontaneous occurrence of nasal tumors in mink at any age. This study supports the concept that NNN is a proven carcinogen for multiple species of mammals and that the mink can serve as a non rodent, non inbred animal model for nasal carcinogenesis, especially since NNN induces only tumors in the nasal cavity in this species and not at other sites, as it does in mice, rats and hamsters.

5/AU,TI,SO,AB/29 (Item 29 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Use of phenylpropanolamine to reduce nicotine cessation induced weight gain in rats.
Winders SE; Dykstra T; Coday MC; Amos JC; Wilson MR; Wilkins DR
Psychopharmacology (Berl) 1992, 108 (4) p501-6,
The present study was conducted to determine if phenylpropanolamine (PPA) administered during the first week of nicotine termination could reduce or eliminate the body weight rebound which accompanies nicotine cessation. Sprague-Dawley rats were administered nicotine for 2 weeks after which they received either PPA or saline for 1 week. Control animals received saline during both drug periods. Body weight, food consumption, and water consumption were measured daily before drug, during nicotine and PPA administration, and for 14 days after PPA administration. In contrast to animals receiving saline upon termination of nicotine, animals receiving PPA did not gain weight at an accelerated rate. Termination of PPA did not result in a body weight rebound. To the extent that these results generalize to humans, they suggest that PPA could be used to reduce or

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eliminate postcessation weight gain in smokers who stop smoking.

5/AU,TI,SO,AB/30 (Item 30 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

National working conference on smoking and body weight. Task Force 2: Methods of assessment, strategies for research.
Henningfield JE; Obarzanek E; Benowitz NL; Hall SM; Kiesges RC; Leischow S; Levin ED; Perkins KA; Spring B; Stitzer M; et al
Health Psychol 1992, 11 Suppl p10-6,

5/AU,TI,SO,AB/31 (Item 31 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Alterations in hippocampal cholinergic receptors and hippocampal behaviors after early exposure to nicotine.
Yanai J; Pick CG; Rogel Fuchs Y; Zahalka EA
Brain Res Bull Sep Oct 1992, 29 (3-4) p363-8,
Mice were exposed to nicotine prenatally by injecting the mother with 1.5 mg/kg nicotine SC twice daily on gestation days 9-18 (PreN mice) or neonatally by daily SC injections of 1.5 mg/kg nicotine on postnatal days 2-21 (NeoN mice). At age 50 days, hippocampal muscarinic receptors Bmax of PreN and NeoN mice were 58% and 79% above control, respectively (p less than 0.01); Kd was unaffected by early nicotine exposure. Eight-arm maze performance of nicotine-exposed animals fell behind control level. Both PreN and NeoN made approximately 10% less correct responses in the first eight trials than controls throughout the test period (p less than 0.01). By the last day of testing, PreN needed 23% and NeoN 31% more trials than controls to enter all arms (p less than 0.001). In addition, PreN needed 35 and NeoN 42% more days than controls to reach criterion (p less than 0.05). Similarly, while 61% of controls reached criterion by day 6 only 17% of PreN and 25% of NeoN reached criterion (p less than 0.01). In the Morris maze, PreN needed from 13 110% more time to reach the platform (p less than 0.001). In the spatial probe test, PreN animals made 35% fewer crosses over the area of the missing platform (p less than 0.001). The study suggests that nicotine administered to the fetus or neonate alters septohippocampal chemistry and induces deficits in hippocampus-related behaviors. The possible reversal of the behavioral changes by manipulating the cholinergic innervations should be the subject of future investigations.

5/AU,TI,SO,AB/32 (Item 32 from file: 155)
DIALOG(R)File 155:(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

Cigarette smoke exposure and development of infants throughout the first year of life: influence of passive smoking and nursing on cotinine levels in breast milk and infant's urine.
Schulte-Hobein B; Schwartz Bickenbach D; Abt S; Plum C; Nau H
Acta Paediatr Jun Jul 1992, 81 (6-7) p550-7,
The effects of smoke exposure via mothers' milk and/or via passive smoking during the first year of life were investigated in a prospective longitudinal matched pair study. The somatic and mental development of 69 infants whose mothers smoked more than five cigarettes per day throughout pregnancy and continued smoking after childbirth were compared with 69 children of non-smoking mothers. At birth, mean body weight of neonates from smoking mothers was significantly lower than the weight of neonates from non-smoking mothers. This weight difference between the two groups was no longer significant in infants at 12 months of age. With the methods

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